PULMONARY TUBERCULOSIS*

THE IMPORTANCE OF THE CLINICAL HISTORY IN ITS DIAGNOSIS

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DISCUSSION *by Philip H. Pierson, M. D., San Francisco; William C. Voorsanger, M. D., San Francisco; A. L. Bramkamp, M. D., Banning.*

EARLY tuberculosis is a curable disease. This is true both of the insidious type and of the type with acute onset. The detail in the method of treating early cases of the insidious type must necessarily differ from that instituted in treating the more acute types. While they cannot all be successfully treated by the same method, yet modern medicine has devised ways by which nearly all of the early limited lesions can be brought to a satisfactory issue, whether the onset be insidious or acute.

Such favorable results, however, can only be attained regularly by the prompt institution of the proper remedial measures before extensive destruction of lung tissue has taken place, and before serious inroads have been made on the patient's resistance; and further, before healing is complicated by insurmountable mechanical problems.

UNDERLYING BASIS FOR CURE IN TUBERCULOSIS

Early diagnosis and immediate adequate treatment is the only procedure which can make tuberculosis a curable disease in the great majority of instances. Delay, while it does not necessarily produce a hopeless condition, as was formerly believed, is nevertheless the one greatest factor which stands between the tuberculous patient and a life of usefulness. This fact must be emphasized until it always stands uppermost in the minds of the doctor and the patient when a diagnosis of early tuberculosis has been made.

The diagnosis of tuberculosis in instances of frank disease is comparatively easy. The knowledge possessed by the well-trained practitioner should be sufficient. It is only in the difficult cases that there should be much doubt.

Many practitioners do not have sufficient experience to become expert in the diagnosis of difficult cases. These will require the opinion of specialists. But careful history taking, as I shall attempt to show in this paper, with analysis of the elicited symptoms, will make the diagnosis fairly certain in nearly all instances of frank disease, and will make the diagnosis probable in a very large percentage of positive border-line cases.

Probably 80 per cent of cases of early clinical tuberculosis can be placed in the class of "probably" or "definitely tuberculous" by the analysis of a carefully taken clinical history alone. This statement is made in face of the fact that tuberculosis does not make itself known in any set way.

The disease, when it becomes sufficiently pronounced to be a clinical entity, is recognized by the fact that it causes disturbances in the body's normal physiologic activity. The clinical history should reveal its course from the time that symptoms first manifest themselves up to and including the time of examination.

SYMPTOMS AND THEIR CAUSES

In order to appreciate the nature of symptoms in tuberculosis one must understand what takes place from the time of infection until clinical disease manifests itself.

Tuberculosis differs from the acute infectious diseases in that the latter, as a rule, consist of one single episode of infection and immunity response, while tuberculosis consists of many such episodes. In the acute infections the patient either dies or develops a more or less lasting immunity to the causative microörganism. A succeeding infection of the same nature is occasionally met, but only rarely. In tuberculosis, on the other hand, the whole clinical course of the chronic disease consists of repetitions of bacillary inoculations and immunity responses with the production of never more than a relative immunity.

In chronic tuberculosis, reinoculations occur in an immunized host and therefore differ from the primary infection. The host being already immunized by previous infection, the immunity response to the reinoculations does not await the usual prodromal stage (which is the period necessary to bring the host's immunizing mechanism into play) but starts at once. If a sufficient number of bacilli engage in the reinoculation, an inflammatory reaction of varying severity depending upon the degree of allergy present is immediately called forth, by which toxins are set free, and pulmonary nerves and local cells are at once irritated, producing disturbances in the host's physiologic equilibrium. These departures from normal physiologic action are recognized as symptoms of tuberculosis. Not only do the symptoms appear sooner, but they are apt to be more pronounced than those due to a primary inoculation, caused by equal numbers of bacilli.

The immediate reaction of the host to primary infection is mild and symptomless. Cells proliferate and attempt to wall the bacilli in, forming tubercles; but there is no general widespread body reaction until multiplication of bacilli with the elaboration and dissemination of tuberculoprotein into the tissues has taken place, and the specific defensive forces of the host have been thereby aroused.

If the numbers of bacilli engaged in reinoculation are few, the reaction will be mild and may be symptomless; but if the numbers engaged in the process are sufficiently large, then recognizable

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TABLE 1.—*Etiological Classification of Symptoms of Pulmonary Tuberculosis*

Group 1 Symptoms Due to Toxemia and Other Causes Acting Generally	Group 2 Symptoms Due to Reflex Cause	Group 3 Symptoms Due to the Tuberculosis Process per se
Malaise Lack of endurance Loss of strength Nerve instability Digestive disturbances (hypomotility and hyposecretion) Metabolic disturbances resulting in loss of weight Increased pulse rate Night sweats Temperature Blood changes	Hoarseness Tickling in larynx Cough Digestive disturbances (hypermotility and hypersecretion) which may result in loss of weight Circulatory disturbances Chest and shoulder pains Flushing of face Spasm of muscles of shoulder girdle Diminished motion of affected side	Frequent and protracted colds (tuberculous bronchitis) Spitting of blood Pleurisy (tuberculosis of pleura) Sputum

symptoms of disease appear within a few hours after the infection has taken place.

ALLERGY IN TUBERCULOSIS

Allergy produces its effects on the host in the following ways: (1) it hastens the elaboration of tuberculoprotein in the focus of infection; (2) it results in the death of both bacilli and tissue cells; (3) through its inflammatory effect on the nerve endings in the tissues it causes reflex symptoms to appear in other tissues and organs; and (4) acting locally, it produces certain recognizable effects in the tissues which are the seat of the lesion.

THREE GROUPS OF SYMPTOMS PRODUCED

These modes of action produce three groups of symptoms, each group having a distinct etiologic cause. The first two produce the toxic group; the third, the reflex group; and the fourth, the local group or those caused by the tuberculous process *per se*. I first proposed this grouping of symptoms in 1913 and have used it continuously in my practice since. I find it very helpful in that it explains what is going on within the lung. It also proves very helpful in differentiating difficult border-line cases.

The important symptoms so grouped appear in Table 1.

ADVANTAGES OF PROPOSED GROUPING

Two important advantages of considering symptoms according to this grouping are: first, that of showing what it is that is operating to disturb the patient's well-being; and, second, through what agencies such disturbance takes place. The allergic inflammatory reaction is responsible for the production of the symptoms in each group.

Nerves, endocrines, electrolytes and cells generally are structures through which the agents act in the production of the symptoms of the toxic group, because they are caused by circulating toxins acting generally throughout the body.

Afferent and efferent nerves and the tissues in the limited area which come under the influence of the efferent impulses cause the reflex group.

The allergic inflammation acting directly on the tissues is responsible for the symptoms of Group 3.

It is evident that the symptoms which indicate the presence of tuberculous disease vary with the dose of bacilli responsible for the reinoculation, the amount of tuberculoprotein which gains access to the circulating blood, the reacting capacity of the patient, and the manner in which the disease progresses thereafter. It is also evident that different degrees of sensitization of body cells result from similar inoculations in different individuals because of the different reacting qualities which are manifested by individuals possessing different cellular reactions.

While bacilli have no power of locomotion within themselves, yet so long as avenues of escape remain for bacilli which are contained within active tuberculous foci, they will gain access to adjacent tissues and through the lymph and blood and natural channels be carried out into other tissues. If the numbers of migrating bacilli are few, and the amount of bacillary protein gaining access to the body fluids is small, reinoculations may take place at frequent intervals without causing any recognizable symptoms; for while they stimulate and act upon the immunizing mechanism qualitatively, the same as large doses of bacilli, or larger quantities of tuberculoprotein, they produce a scarcely perceptible effect quantitatively. The resulting allergic reaction may be so slight as to be microscopic and so, of course, produce no recognizable symptoms; or, it may be more marked and still not be discernible; or, it may be so severe as to precipitate a marked defensive response on the part of the host with a toxic syndrome comparable to that which accompanies the acute infections. In fact, such a reinoculation is accompanied by the same episode of immunity response as characterizes such diseases as diphtheria, scarlet fever or measles.

Again bacillus bearing discharges which are cast off into such natural channels as the bronchi may plug the same and thus cause a retention of bacilli *in situ* until they have initiated an infection. Such an infection as a rule would produce an abrupt onset of symptoms. We do not believe this occurs often in the beginning of pulmonary disease except following the rupture of a caseating bronchial gland, yet we must accept it as not an uncommon possibility in the extensions which

take place from pulmonary foci to unaffected tissue during the course of advanced tuberculosis.

There is probably a period in all early active tuberculous infections, either before or after they have made themselves known by frank symptoms, when bacilli are carried in minimal numbers through the body fluids from existing foci to new tissues, and when tuberculoprotein circulates in the body fluids in minute quantities. The resulting reaction may be so slight that it produces no recognizable disturbances in the physiologic equilibrium of the host. No doubt, many borderline cases which react markedly and quickly to the cutaneous and intradermal application of tuberculin in the presence of indefinite and inconclusive symptoms and thus puzzle the examiner in forming a conclusion as to whether or not active tuberculosis is present, belong to this class. They are potentially tuberculous but may not become actually clinically ill unless larger reinoculations take place. A clinical history in such cases is not conclusive. Further evidence must be found on which to base a diagnosis.

Frequently repeated reinoculations, too, may be caused by larger numbers of bacilli; and, larger quantities of tuberculoprotein may escape from existing foci, and still the reaction not come within the domain of distinct acute inflammation with its marked toxic and reflex symptoms, such as characterize the acute infections. The patient may have a slight elevation of temperature, a loss of vigor, fatigue, possibly lack of appetite and loss of a few pounds in weight, yet be unable to point to a definite episode of immunity reaction such as would characterize an acute allergic response.

The pathology in these cases consists of slight inflammatory phenomena, but so slight that they may be detected only with difficulty by the usual procedures of physical examination, or by the x-ray, except after a tuberculin reaction of sufficient magnitude to change the mild allergic reaction of a predominantly proliferative character to one of a predominantly exudative (parenchymatous) character.

Tuberculosis of this type in an active form may be present for quite a period of time before it causes sufficient symptoms to make a diagnosis definite; in fact, may heal before causing sufficient symptoms to make the diagnosis definite.

On the other hand, when tuberculosis shows itself as a frank disease, with a marked immunity response accompanied by an acute toxic reaction there nearly always will be reflex phenomena present, and often, too, evidence of the local reaction of the disease in the tissues such as sputum, a pleural involvement or an hemoptysis, to make the diagnosis quite evident.

It is necessary for the profession to know that the disease may come on insidiously with small reinoculations and no frank symptoms, or abruptly with acute toxic mainfestations, for much of the teaching in the past has not taken this sufficiently into consideration.

It is very desirable but quite impossible to assign definite values to the different symptoms. This is impossible because different people react differently to the same stimulus; and, further because different organs in the same individual may show differences in their response; and, still further, because the reinoculating doses of bacilli are variable in size and virulence.

DIAGNOSTIC VALUE OF SYMPTOM GROUPS

The three groups of symptoms vary greatly in their diagnostic value. The toxic or general group is characterized by the fact that it represents harmful influences which affect structures throughout the body; nerves, endocrine glands, and body cells. The symptoms which accompany the acute reaction following a reinoculation with fairly large quantities of bacilli, is qualitatively the same as that which follows reinoculations of milder degree, and similar to the symptoms which accompany neurasthenic and psychasthenic states or conditions of hypo- or hyperactivity in certain endocrine glands, such as the thyroid, gonads and adrenals; but they differ in severity. Nor does the acutely toxic state in tuberculosis differ in symptomatology from the acutely toxic state in other infections; so there is nothing significant or of differential diagnostic import in the symptoms of Group 1. They must be combined with symptoms of Groups 2 and 3 to possess diagnostic worth. They only show that some factor or factors are producing a widespread injury to the body tissues and functions.

Symptoms belonging to the reflex group, on the other hand, possess considerable diagnostic value, even on their own account. Irritation of the larynx, hoarseness and cough are one or all usually present in early clinical tuberculosis, but they do not possess so great localizing worth as the reflex spasticity which involves the skeletal muscles; for the cough reflex may be produced by stimuli arising in many tissues other than pulmonary. The value of the reflex symptoms is greatly increased by the fact that some symptoms of Groups 1 and 3 are nearly always present at the same time, or, if not present, there is a history of their presence in the near past; and the combination of the symptoms of the two or three groups is decidedly more suggestive than those of one group alone.

When the lung is the seat of allergic reaction, as it always is in active pulmonary tuberculosis, stimuli are carried to the central nervous system over the visceral nerves; and transferred to those muscles which receive their innervation from the midcervical segments of the cord, causing them to show reflex spasticity. This may be detected as an increased tension and as an uneven contracture of the muscle bundles on palpation, and may also be inferred from the lessened motion of the hemothorax corresponding to the lesion if it is

one-sided, or, from the detectable asymmetrical movements when both sides are involved. Lagging thus when properly evaluated becomes an important sign of active pulmonary inflammation.

The structures involved are the sternocleidomastoideus, scaleni, pectorales, subclavii, trapezii, levator anguli scapulae and rhomboidei muscles, and the crura and central tendon of the diaphragm.

This spasticity and the effects which it exerts is of the greatest diagnostic worth and when combined with subjective symptoms of a reflex or toxic nature immediately fixes attention upon the lung because of its definite localizing nature.

Flushing of the face is also of value, but we rarely see it as one of the early symptoms. It is more apt to appear after the lung has been the seat of disease for some time.

The symptoms of Group 3 are of the greatest diagnostic import. They are subjective symptoms about which the patient will tell you. They have no more direct localizing value than the spasticity of the muscles, but they are complained of while the latter must be detected by the examiner.

Pleurisy, hemoptysis and scanty sputum are commonly present in early or fairly early tuberculous lesions. If the sputum contains bacilli, the diagnosis is made; but it is sometimes present in small amounts in early lesions without bacilli being found in it after most careful search.

Hemoptysis may be met in influenza, and post-influenzal infection, bronchiectasis, lung abscess, lung syphilis and malignancies; but the history will usually suggest whether or not these other conditions are present. If symptoms of both Groups 1 and 2 are present with an hemoptysis of one-half to one dram of bright blood, it points most strongly to tuberculosis as the causative factor, and at least calls for a longer observation and a carefully directed plan of living.

Pleurisy, except it accompany influenza, pneumonia, or an injury to the chest, is nearly always due to tuberculous infection.

SUMMARY

With the understanding that there is only one etiologic factor responsible for the symptoms of early clinical tuberculosis, viz., the allergic reaction; and with the further knowledge that this causes the symptoms in one of three ways, through toxins, reflexly, or locally at the point of the inflammation, we are now able to see the combined value of symptoms of at least two or of all three groups in fixing the diagnosis of a given pulmonary infection.

Since the allergic reaction is also responsible for the evidence found on physical examination and that revealed by the x-ray, it may be seen that the reaction which causes few symptoms is also likely to cause few signs which are demonstrable on physical and x-ray examination. It is in such cases that we need help from all the diagnostic methods that we have at our command. This paper is not intended to belittle other methods of examination, but simply to call attention to and to emphasize the importance of a clinical history, when accurately taken and carefully analyzed.

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DISCUSSION

PHILIP H. PIERSON, M. D. (490 Post Street, San Francisco).—This paper dealing with the clinical history in the diagnosis of pulmonary tuberculosis is very timely. Today when there are so many short-cuts to diagnosis by means of laboratory aids, the careful taking of the history is often neglected. It has been my custom to ask the patient how long ago he was *perfectly well, active and strong* and chronologically put down the symptoms as they have occurred, very often over several years. One of the most important groups of allergic phenomena is the gastro-intestinal group mentioned under one and two. Often suggestive of chronic disturbance of the gall bladder, appendix, or colon, much time is lost in treating the patient for an illness which is not really responsible for his complaint. Likewise the histories of frequent colds are merely allergic reactions about a pulmonary or bronchial focus. This is particularly true in childhood. Among other helpful aids in determining their sensitiveness of tuberculosis is the intracutaneous tuberculosis test. This reaction is often more marked at one time than another. Serial roentgenograms frequently bear out this changing reaction about a pulmonary focus.

In group two, Doctor Pottenger has mentioned spasm of muscles of the shoulder girdle as a reflex manifestation of trouble in the lungs. In early tuberculosis, especially in the face of hemorrhage where a thorough examination is impossible, I am sure we can gain much from palpating the chest to determine lagging of one or the other side and this reflex spasticity of the muscles reflecting the underlying disease. To be sure it takes a good deal of experience to properly interpret this sign, but I feel that it is well worth special attention in order that we may be acquainted with it accurately when it is most needed. The old adage, treat the patient and the disease will get well, is particularly true in tuberculosis and the recognition of the many allergic phenomena, as expressed by Doctor Pottenger, will help in choosing the proper system of treatment for tuberculosis.

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WILLIAM C. VOORSANGER, M. D. (490 Post Street, San Francisco).—The diagnosis of beginning tuberculosis depends largely upon symptoms elicited, gathered only by careful questioning of the patient. As the doctor so well states, our hope for cure lies in starting remedial measures before the tuberculous process has made too great inroads into the lungs. The specialist, too, seldom sees the beginning disease—too often, sad to relate, the patient comes to him with advanced tuberculosis which has remained undiagnosed. Careful history-taking will often elicit a slight cough of months' duration, pains in the chest, a pneumonia or influenza in previous years, a steadily growing loss of appetite, an occasional night sweat—but, most important of all, a definite statement of fatigability of which the patient himself may have been ignorant until it is called to his attention. While these symptoms do not always indicate tuberculosis, they are highly suggestive of it, and if kept in mind, will lead the physician to make a complete examination, with sputum analysis and x-ray investigation.

Without a careful history, and without properly evaluating elicited facts, the patient is often dismissed with a little advice or a cough mixture and thus permitted to lose his chance of getting well. I agree

fully with Doctor Pottenger concerning repetitions of bacillary inoculations in tuberculosis and that we only accomplish a relative immunity. Regarding the primary infection, we have learned that it does not always start in the apex as formerly believed; it starts most often infraclavicularly, and an early lesion may thus be overlooked by the ordinary physical examination.

Doctor Pottenger's grouping of symptoms is excellent; you will notice the most distressing ones are due to toxemia, which can only be combated by rest in bed. How necessary, therefore, to make an early diagnosis and get our patients at rest!

Time will not permit lengthy discussion of the statement that the allergic reaction is the main etiological factor in pulmonary tuberculosis. We are beginning to recognize this fact in other diseases, and particularly in other pulmonary conditions; perhaps it explains why we have so often failed to effect a cure in one patient while accomplishing it in another. It is certainly a true and important statement, if reactions in the human body can cause mild symptoms in one person, and severe ones in another, it surely becomes self-evident that a careful eliciting of all facts which can have a bearing upon an early diagnosis is a matter of the first importance.

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A. L. BRAMKAMP, M. D. (Banning).—For many years, in season and out of season, Doctor Pottenger has been preaching to medical men this gospel of the curability of pulmonary tuberculosis based on early diagnosis and treatment.

On the whole it may be accepted as a fact that doctors generally are now somewhat better able to recognize the clinical disease from physical signs than formerly if serious and persistent effort is made. However, in many cases, the disease will have done considerable damage in the lungs by the time physical signs are readily detectable. We need to be "tuberculosis minded," always alert to the possibility of its existence even in the apparently well or slightly indisposed.

While it is true that other diseases are accompanied by many of the symptoms of pulmonary tuberculosis of the toxemia group, if the toxemic symptoms in a particular case are accompanied also by those of the reflex and focal groups the evidence is so compelling that we should consider the case one of tuberculosis until some other fully adequate explanation is found.

Just as in years past, moderately or far-advanced cases form the great majority of patients in sanatoria. Many of these patients have had relatively early diagnosis and therefore are perhaps themselves responsible for their failure to recover. Since the change to the present hopeful attitude as to the curability of the disease, there is lessened stigma attached to those who have it. And particularly, since the patient's own efforts and coöperation are such large factors in determining the outcome, can there be any justification for failure to inform the patient early and fully as to the diagnosis.

It is well to keep in mind that pulmonary tuberculosis in children and adolescents is more common than formerly realized; that in these young people (as in some adults) physical signs of the disease may be very indefinite or altogether lacking. In these patients the clinical history may have to be relied upon almost wholly. Fortunately in these cases the x-ray often affords definitely corroborative evidence.

Doctor Pottenger's emphasis on the value of an adequate history as a factor in the early diagnosis of pulmonary tuberculosis, even before substantiating physical signs are present, is as important and as timely as ever.

THE TEACHING OF PERINEAL PROSTATECTOMY*

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DISCUSSION *by Ralph Williams, M. D., Los Angeles; R. L. Rigdon, M. D., San Francisco; Robert V. Day, M. D., Los Angeles.*

THE operation of "conservative perineal prostatectomy" holds a rather unique position in the field of surgery. It has passed through several short periods of popularity alternating with those of marked disfavor. Few surgeons today perform prostatectomy by way of the perineum and it is a matter of some curiosity to the many who do not, why this small group persists in performing perineal prostatectomy. There are two factors that contribute to the disfavor of this operation. One of these is the so-called "median perineal prostatectomy" with which it has often been confused. At the outset it must be recognized that Young's conservative perineal prostatectomy is the only safe perineal operation for removing enlargements of the prostate and when properly performed is a highly technical surgical procedure, whereas median perineal prostatectomy is a blind, unsurgical method, unworthy of comparison. The results are in no sense comparable. Another factor that has contributed largely to the disfavor of conservative perineal prostatectomy is the fact of its having been attempted in the past by men unprepared to perform it. It must be recognized that the operation can be performed successfully in one way and one way only, so far as fundamentals are concerned, and this one way was first outlined by Young. Modifications that have since appeared are of relatively minor importance. The Young method preserves the rectum and the external sphincter and the ability properly to do this is the stumbling-block of the operation.

THEORETICAL ADVANTAGES OF PERINEAL PROSTATECTOMY

The theoretical advantages of perineal prostatectomy over suprapubic prostatectomy are numerous. Regional anesthesia is much more satisfactory by way of the perineum. Complications and dangers of infection are much less, the perineum having apparently a localized immunity which the suprapubic route lacks. Furthermore, the suprapubic incision, because of the proximity and danger of injury of the peritoneum and because of the complications that arise from infections of the space of Retzius or the perivesical regions, produces marked postoperative burdens that the perineal route escapes. Keyes, recognizing this danger from infection, has advocated suprapubic prevesical section, the bladder not to be opened until after it has become adherent to the edges of the suprapubic wound so as to prevent spread of infection. But the suprapubic route rivals perineal surgery only when the open,

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